

## Obesity treatment for adults, adolescents and children

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Obesity treatment aims to produce weight loss and weight-loss maintenance, as well as risk reduction, disease prevention, amelioration or cure. Treatment needs to be designed according to the person's need. A small loss (5%–10% of initial body weight) may suffice for disease prevention, but greater weight loss is often needed for mobility or for disease cure in those with a greater degree of obesity. For children and adolescents, family involvement and a developmentally appropriate approach are vital. For example, for pre-pubertal children, who are still growing in height, reduction in the rate of weight gain, or weight stability, may be sufficient to achieve health improvement. Lifestyle interventions to achieve energy restriction and increase energy expenditure (through activity and exercise) are the initial approach. Pharmacotherapy can improve the efficacy of lifestyle interventions. Intra-gastric devices and bariatric surgery may be appropriate for those with more severe disease, including selected older adolescents. While weight loss can be achieved, the major issue in treatment of obesity remains weight maintenance and efforts need to be directed to this end.

Overweight and obesity are common in Australia, with approximately one in five adults being obese, two in five overweight and approximately one in four of our children being overweight or obese. This condition has essentially become the norm. As is discussed elsewhere in this volume, this increasing obesity prevalence has come with increasing health problems and diseases. Whilst many of these diseases can be treated directly (and at continuing expense) it would be better to prevent such obesity-associated diseases by treating their underlying cause, that is by managing overweight and obesity properly. In fact, it would be even better to prevent overweight and obesity occurring in our society!

Traditionally the health professionals have been pessimistic about their ability to treat obesity but with the new realisations of what successful treatment really is, and with the newer treatment modalities, obesity treatment can be, and is, successful. What is needed

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is a practical approach, individual goals for each patient and a system which provides the appropriate intervention as well as continuing support and follow-up. These aspects of obesity management for adults, adolescents and children will be discussed in the chapter.

### Management of obesity in adults: whom to treat and how to treat?

There are a number of modalities involved in the management of obesity, which should be delivered as an integrated program. These modalities include standard lifestyle interventions (eating and activity), behaviour and group therapy, adjunctive therapy (pharmacotherapy, meal replacement regimes), some special devices (intra-gastric balloons) and bariatric surgery. A suggested approach utilising the patient's body mass index (BMI, weight/height<sup>2</sup>, kg/m<sup>2</sup>), the presence of obesity-associated comorbidities, and a range of treatment options is outlined in Table 1.

Table 1. A management approach for obesity in adults.

BMI (kg/m <sup>2</sup> )*	Lifestyle**	Obesity drugs	VLEDs	Devices: eg intra-gastric balloon	Bariatric surgery
25 to 26.9	Advice				
27 to 34.9	Lifestyle program	Consider	Possibly		
27 to 34.9 ±comorbidity	Lifestyle program	Consider	Probably	Possibly	
>35 ±comorbidity	Lifestyle program	Consider	Utilise	Possibly	Consider
>40 (if no comorbidities)	Lifestyle program	Consider	Utilise	Possibly	Consider

\* If there is increased waist circumference (the importance of which is described elsewhere in this volume) then a more intensive therapeutic regimen may be utilised at a lower BMI range.

\*\* All the components of the lifestyle program and other therapies are described below. Obesity pharmacotherapy may be utilised to help maintain weight after it is lost using other treatment modalities instead of just being used to produce weight loss. Very low energy diets (VLEDs) are useful for initiating and obtaining weight loss, in weight maintenance and prior to bariatric surgery.

It is important that healthcare practitioners recognise when a patient has a weight problem and for them to be able to prescribe an appropriate management plan for the patient's degree of obesity, life stage and situation.

### Lifestyle management

A lifestyle-management program remains the basis of all obesity treatment [1]. Such a program has a number of components, the most obvious of which are eating and physical activity plans. Another major component is a behaviour-modification program. Other

aspects include goal setting, psychological support (and counselling if necessary), proper medical management and a long-term plan for maintaining weight loss. Such lifestyle programs have been shown to be effective [2], and losses of 6% or more of initial body weight can be achieved and maintained. There is no reason for such programs to be delivered solely by health professionals [3]. For those with a BMI in the lower overweight and obesity range (ie BMI 27 to 34.9 kg/m<sup>2</sup>), and without major medical complications, such programs can be delivered effectively in the community setting.

### Goal setting

This is often ignored, or not specifically discussed with the patient. The goals will vary from patient to patient. It is important to set realistic goals for weight loss, of course, but other goals may be individual and/or medical. Other than weight loss, goals may include prevention and control of disease (such as diabetes), better mobility, less medications, ability to have a joint replacement, or a specific goal that the patient wants to achieve. Such goals should be discussed, recorded and when reached the achievement should be noted and the patient congratulated.

### Eating

It is better to use the term 'eating plan' rather than 'diet'. Diets tend to be seen as short term and there are many which are pushed by magazines, in books, and online, and there is a large industry producing these diet plans. This plethora of diets is confusing and unhelpful for someone attempting to lose and then maintain weight loss. It has been shown that the amount of weight loss obtained does not depend on the type of eating plan prescribed; most produce similar weight losses at six months with gradual weight regain thereafter [4, 5]. Compliance to the plan, not the type of plan, is what produces weight loss [6]. What is really necessary is a 500 to 600 kcal (2100 to 2500 kJ) energy deficit and a plan that starts by making small changes to a patient's habitual consumption. Such changes may include decreases in portion size, a reduction in fat (and in particular saturated fat), possibly an increase in mono-unsaturated fat, a reduction in sugar-sweetened beverages and a low glycaemic index diet. An increase in protein in the diet may increase satiety. Any changes must be made specifically in the context of the patient's habitual diet. It is useful to keep food logs at regular intervals to check compliance and to use as a tool to suggest further changes in intake or eating patterns.

### Physical activity

The term 'physical activity plan' is preferred to 'exercise', because the prescribed plan will include both planned exercise as well as incidental activity. A formal prescription of such a plan does produce greater compliance. Activity helps maintain lean mass and because of the increased energy deficit it promotes weight (kg) and fat mass loss. A simple initial approach is to use a physical activity questionnaire, determine how much time is spent walking or in specific exercise and perhaps suggest the use of a pedometer to get an activity baseline.

The activity prescription itself should be specific to the patient. It may include increased time walking, more incidental activity (taking an active option), and/or specific exercises, gym programs or training, or involvement in sport. For diabetes prevention, supervised exercise sessions have been shown to be part of all-effective programs. It is good to remember that the greater the patient's weight, the fewer 'steps' they have to do to lose weight. Activity should be monitored and the amount done recorded. Again this will help in assessment of compliance and will also be a tool for increasing the amount of activity performed.

Increased activity alone (as a weight-loss program) does not produce a great deal of weight loss. It may affect fat distribution [7, 8] but, for reasonable weight loss to ensue, activity must be combined with an eating plan [9].

#### Behaviour modification

The use of food and activity logs is part of behaviour modification. Another log that may be useful is one for mood, particularly at time of eating, as well as general affect. Cognitive behaviour therapy may be useful, particularly for optimising weight maintenance. Other techniques are those of cognitive restructuring, motivational enhancement therapy, psychological support and counselling. There are many techniques that may be used but to describe these in any detail is beyond the scope of this chapter. The techniques utilised need to be individualised to the patient's situation and life stage, so there really is no 'standard' program – rather, a series of techniques used when necessary [10].

An effective lifestyle program must contain some or all of these elements, and though patient education alone is not effective in producing weight loss, it too is a part of a successful program. A multidisciplinary team providing care is essential. Regular visits (at least fortnightly initially and then at longer intervals) are important. Weight regain does tend to recur, particularly in the second year of a program and it is at this stage that there needs to be further research on the effect of repeated, brief acute interventions, the use of pharmacotherapy for maintenance, and/or the use of very low energy diets (VLEDs).

If the lifestyle program is not successful, with goals not being attained, then adjunctive therapy should be considered. Such consideration should begin early in any program, say at one month, with reconsideration at regular intervals.

#### Adjunctive therapy

##### *Very low energy diets (VLEDs)*

Such formulations are effective at producing early weight loss [11], although after a time (two to three years) they appear to produce no more weight loss than a standard lifestyle program. However if their use is continued (as one to two meal replacements per day) then significant weight loss can be both achieved and maintained [12]. These diets tend to contain 500 to 800 kcal/day and the necessary vitamins and micronutrients. Early formulations did not contain high-quality protein and a number of deaths ensued, but these difficulties have been addressed and current formulations are safe, provided they are used according to protocol, and preferably under medical supervision [13].

These formulations are available as shakes, soups and bars, and in most countries they are classified as 'Foods'. They may be used as a full meal replacement protocol (every meal a VLED) or as a partial protocol (one to two meals a day as a VLED). The full protocol is best performed under clinical supervision, particularly if the patient has diabetes or cardiac disease. The full protocol has been used as the 'control treatment' in randomised trials of bariatric surgery and quite reasonable weight losses, of the order of 15% or more of initial body weight, are obtained.

Initial full-meal replacement protocols usually run for 12 to 16 weeks though under supervised conditions there is no reason why they should not be used for longer. They can be used as one to two meals per day to help maintain weight loss. They are generally used prior to bariatric surgery [14] to reduce some weight and to reduce fat in the liver (which makes surgery easier).

### *Pharmacotherapy*

The relatively limited success of lifestyle interventions has driven the search for effective anti-obesity drugs. Drug treatment of obesity is driven by similar principles for pharmacotherapy of other chronic diseases: it needs to be effective and safe (particularly as it will need to be used long term), acceptable to patients and affordable [15]. While the physiological control of body weight theoretically provides many targets (satiety, nutrient absorption and energy expenditure), in practice it has proven hard to find safe and effective drugs and regulatory authorities such as the Food and Drugs Administration in US, European Medicines Evaluation Agency and Therapeutics Goods Administration in Australia, have set guidelines and standards of evidence for efficacy and safety that have proven a barrier to new drug development.

### *Drugs available in the past 20 years but no longer licensed*

Amphetamine-like drugs with predominant actions of enhancing brain dopamine pathways included phentermine and diethylpropion. They produced anorexia (rather than satiety) and had some CNS stimulant properties. Although they produced weight loss of about 5%–10% they were not evaluated according to contemporary standards for clinical trials. Phentermine is available and used short term in the US, Asia and Australia, but not in Europe. The fenfluramines developed in the 1980s were effective satiety-enhancing drugs that acted mainly through enhancing central serotonergic neuronal transmission (by both release and reuptake inhibition of serotonin). However they were associated with pulmonary hypertension and cardiac valve abnormalities (especially when combined with phentermine) and withdrawn from use in 2004. The endocannabinoid receptor (CB1 receptor) was identified as an attractive target since it had long been known that stimulation of this receptor led to hunger; a number of selective CB1 receptor blockers were developed in the early 2000s, with rimonabant being licensed in Europe on the basis of four clinical trials that showed it could produce weight loss of about 10% absolute, 5.6 kg placebo-subtracted, and that efficacy was maintained over at least two years. Furthermore rimonabant use was associated with metabolic improvements of lipids and glycaemic control. However shortly

after approval it became clear that there was an unacceptable incidence of depression and suicidal ideation with the drug that led to its withdrawal [16].

Sibutramine, a selective serotonin and norepinephrine-uptake inhibitor had been licensed and in use worldwide since the 1990s. It produced placebo-subtracted weight loss of about 5% over one year, but appeared to have greater efficacy when combined with intensive lifestyle interventions: in one trial an average 12.1 kg weight loss compared to five kilograms with sibutramine alone. Concerns remained that the drug had unwanted and potentially dangerous sympathomimetic effects that could raise blood pressure and pulse, so at the demand of the European Medicines Agency the Sibutramine Cardiovascular Outcomes (SCOUT) trial assessed the efficacy of sibutramine in reducing myocardial infarction, stroke, and cardiovascular mortality in 11 000 obese and overweight patients. In order to produce enough cardiovascular endpoints, patients with existing cardiovascular disease and diabetes and at high risk for future events were recruited (a population that would have been excluded according to the drug licence). The trial reported that despite well-sustained weight loss, there was an excess of non-fatal cardiovascular events in the subjects receiving sibutramine for up to four years [17] and this led to the withdrawal of the drug.

#### *Current drugs licensed for obesity treatment*

In 2011 orlistat, a pancreatic and intestinal lipase inhibitor, was the major drug licensed for obesity treatment. It produces malabsorption of about 30% of ingested dietary fat. Its use is limited by gastro-intestinal side effects (steatorrhea, flatus, urgency of defecation and sometimes even faecal incontinence) such that unless subjects are on a low-fat diet (maximum 90 gm fat daily) it is unlikely to be tolerated. In this setting the drug can produce an approximate 300 kcal/day energy loss through undigested fat. In a meta-analysis of 11 placebo-controlled trials of one year in over 6000 overweight or obese patients, orlistat 120 mg thrice daily reduced weight by about 3% more than placebo, with 21% reaching a 5% loss, and 12% a 10% or greater weight loss. Orlistat has favourable effects on blood pressure, lipids and blood glucose. In some countries orlistat is available at a dose of 60 mg thrice daily either over the counter or through pharmacist prescription [18–20].

Phentermine is available in Australia, the US and Asia. It is an older drug with a central action on appetite. There are no long-term studies, but it has been shown to be effective short term and may be used to help produce or maintain weight loss. Further studies are really needed to define its place in obesity management.

#### *Drugs producing weight loss licensed for other indications*

The glucagon-like peptide 1 (GLP-1) incretin analogues exenatide and liraglutide are both licensed for use as hypoglycaemic drugs for treating type 2 diabetes, and they produce a weight loss of two to four kilograms. GLP-1 agonists have complex modes of action on weight loss that include delayed gastric emptying, and both direct, and indirect via the vagus nerve, stimulation of central nervous system satiety pathways. The drug has to be given by subcutaneous injection. Liraglutide given at higher doses than used to treat diabetes is in clinical trials as a weight-loss agent in non-diabetics. Early results have shown

body weight losses of about 10% associated with marked metabolic and cardiovascular improvements sustained for two years [21].

Currently much focus is on combining drugs at low doses to produce synergistic weight-loss effects while minimising side and unwanted effects [22]. Combining naltrexone (an opioid receptor antagonist) with bupropion (an antidepressant) showed synergistic effects on the firing of hypothalamic pro-opiomelanocortin neurons in mice. In a trial of nearly 2000 overweight and obese subjects the highest dose combination produced weight loss of 6.1% at one year compared to 1.3% on placebo. Side effects were mainly gastrointestinal, but there were transient increases in blood pressure with less fall than might have been expected from the weight-loss achieved [23].

The anti-epilepsy drug topiramate has been combined with phentermine and also investigated in clinical trials. In nearly 2500 patients the highest dose combination produced a weight loss after 56 weeks of just under 10% compared to 1.2% of those on placebo. Dizziness, depression and anxiety were the most common side effects and will need further evaluation before the drug will be licensed.

### *Bariatric surgery*

Bariatric procedures aim to produce and maintain weight loss by altering energy balance primarily by reducing food intake and mitigating the physiological changes that drive weight regain. Most guidelines recommend that bariatric surgery be considered for those with a BMI above 35 kg/m<sup>2</sup> with an obesity-related comorbidity, or above 40 kg/m<sup>2</sup> in those without. A multidisciplinary team including surgeon, anaesthetist, physician, dietitian, specialist nurse and psychologist or psychiatrist is needed to provide optimal care.

Four procedures are most commonly performed, in modern practice all laparoscopically: Roux-en-Y gastric bypass, adjustable gastric banding, sleeve gastrectomy and biliopancreatic diversion (Figure 1), with considerable variation between countries in the preferred procedures.

Roux-en-Y gastric bypass creates a small gastric remnant that empties directly into the transposed jejunum, bypassing the main body of the stomach, duodenum and proximal jejunum. Although the procedure was initially thought to produce weight loss by both restriction and malabsorption of food intake, it is clear that the impact of food entering rapidly into the distal jejunum is associated with the rapid release of gastrointestinal peptide satiety hormones including GLP-1 and Peptide YY (PYY), as well as decreasing the orexigenic hormone ghrelin, released from the stomach. Other factors postulated to produce weight loss include altered bile salt secretion and altered gut microbiota. Patients require lifelong vitamin B12 and vitamin D replacement. Weight losses average 35%–40% at five years.

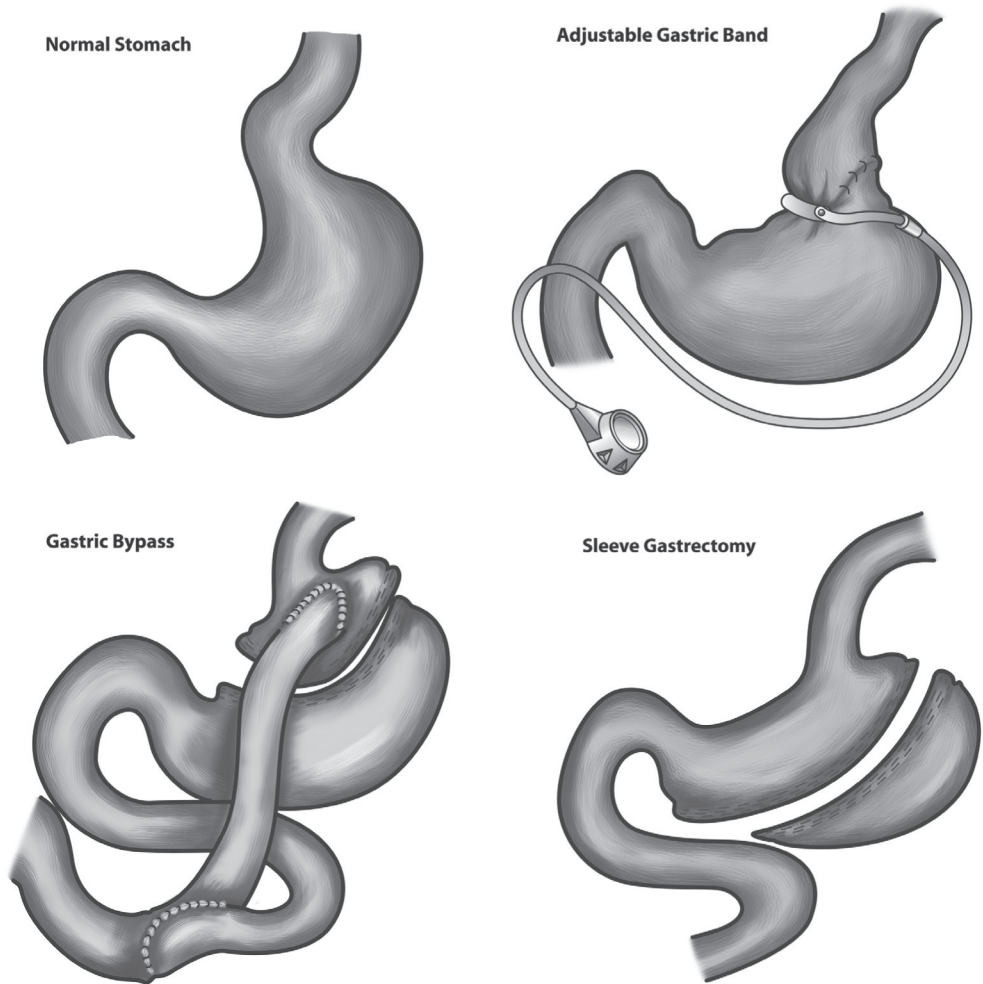


Figure 1. Types of bariatric surgery, shown schematically from top left to bottom right: normal stomach, adjustable gastric band, gastric bypass, sleeve gastrectomy.

Adjustable gastric bypass is a simpler procedure in which an adjustable silastic band is placed around the proximal fundus of the stomach to leave a pouch of about 15 to 30 ccs. A tube leads from the band to a port positioned subcutaneously through which saline can be injected to tighten (or loosen) the band. Frequent adjustments of band filling are required and success is critically dependent on this process as well as the patient's willingness and ability to adapt their diet and respond to the sense of fullness that the band produces. Weight losses average 25%–30% at five years. Sleeve gastrectomy was originally introduced as a first step for patients at too high a surgical risk for a Roux-en-Y procedure, with the intention to convert the gastrectomy to a bypass at a second operation after the patient had lost some weight. The finding that sleeve gastrectomy led to similar weight loss and metabolic benefit as the full Roux-en-Y procedure led to enthusiasm for this technically



simpler procedure, but more recent evaluation suggests weight-loss and metabolic improvement are less durable.

Bilio-pancreatic diversion with or without a duodenal switch procedure is a major malabsorptive procedure that is not widely performed and has been less well evaluated than other procedures. Although weight loss may be 40%–60%, nutritional complications are frequent.

The most compelling data for the success of bariatric surgery at producing weight loss and improving the clinical outcomes for obese patients comes from the 15-year follow-up data of the Swedish Obese Subjects study (SOS) [24]. This was a case controlled study, and the surgical techniques were not as advanced as performed nowadays. The persistent weight loss in the surgical groups was associated with a much reduced mortality: the unadjusted overall hazard ratio was 0.76 in the surgery group ( $P = 0.04$ ), as compared with the control group, and the hazard ratio adjusted for sex, age and risk factors was 0.71 ( $P = 0.01$ ). Other studies have confirmed that bariatric surgery is associated with reduced all-cause mortality including deaths from cardiovascular disease, diabetes and cancer.

Bariatric surgery has important benefits in patients with type 2 diabetes that extend beyond weight loss [14]. Up to 80% of people with type 2 diabetes may experience remission of their diabetes (normoglycaemia without the need for hypoglycaemic medication), the exact remission rate being determined by the type of surgery and the duration of diabetes prior to surgery. Systematic reviews suggest that about 60% will have remission after gastric banding (and that the remission is of slower onset and more closely related to weight loss) compared to 80% after Roux-en-Y bypass, and 95% after bilio-pancreatic diversion. The International Federation of Diabetes position statement in 2011 concludes that bariatric surgery: a) constitutes a powerful option to ameliorate diabetes in severely obese patients, often normalising blood glucose levels, reducing or avoiding the need for medications and providing a potentially cost-effective approach to treating the disease; b) is an appropriate treatment for people with type 2 diabetes and obesity not achieving recommended treatment targets with medical therapies; c) should be an accepted option in people who have type 2 diabetes and a BMI  $>35$ ; and even d) an alternative treatment option in patients with a BMI between 30 and 35 when diabetes cannot be adequately controlled by optimal medical regimen, especially in the presence of other major cardiovascular disease risk factors [25].

Though it is effective for weight loss, the complications of bariatric surgery are significant [26]. The mortality risks of surgery itself are  $<0.5\%$  overall, although the risks for those individuals with pre-existing cardiovascular disease or other morbidity may be substantially higher. Short-term complications are substantially lower with gastric banding compared to other procedures, but later complications are common. Re-operation rates for 'slipped' bands or erosion of the band through the stomach wall may occur in as many as 30% of banded patients after five years and a third may fail to lose or maintain weight loss [27]. Other longer-term complications in gastric bypass patients include strictures, excessive weight loss, and hypoglycaemic dumping. The latter appears to be driven by excessive incretin, and hence insulin responses to (inappropriate) high-carbohydrate meals.

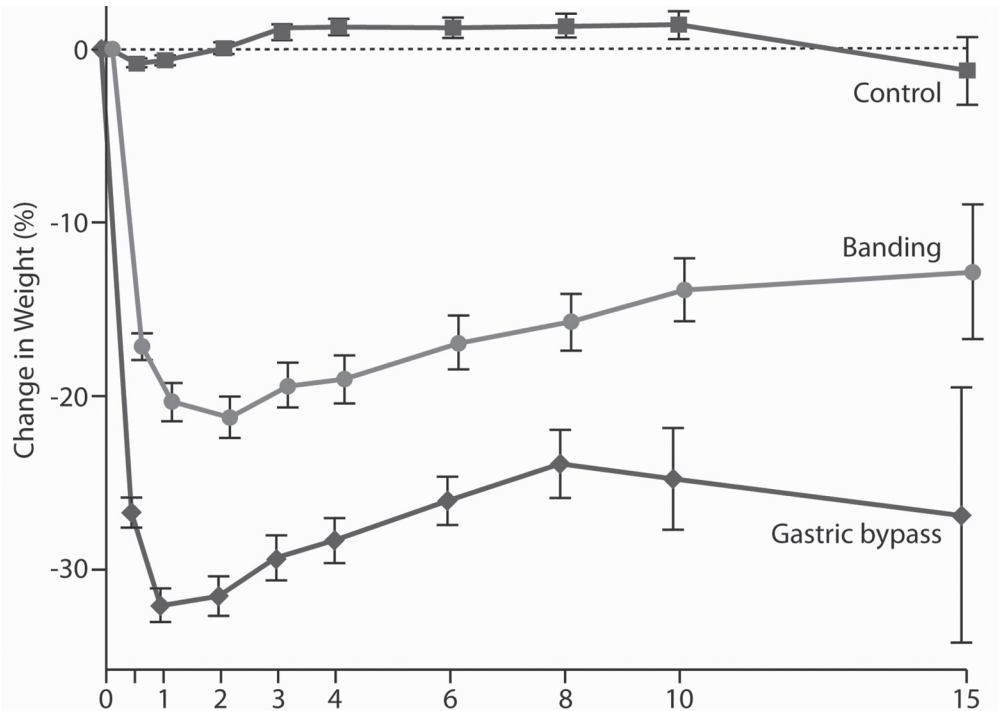


Figure 2. Weight trajectories from Swedish Obese Subjects study. Source: Sjöström L, Narbro K, Sjöström DC, Karason K, Larsson B, et al. (2007). Effects of bariatric surgery on mortality in Swedish obese subjects. *The New England Journal of Medicine*, 357(8): 747.

While bariatric surgery has much to offer, for optimal results highly experienced surgeons doing high volumes of surgery, in patients who have an adequate understanding that surgery is an aid not a 'quick fix' to weight loss, and with excellent nutritional support and more general psychological counseling are needed.

#### Devices

There are a number of devices which have been used to manage obesity. One in relatively common use is the intragastric balloon. This is placed endoscopically, inflated with fluid, and for the first week after placement the patient stays on VLEDs until the balloon becomes fixed. This device restricts eating and in a recent trial with obese subjects we have shown weight losses of 14% of initial body weight at six months compared to 4.8% in controls who were treated with a lifestyle program [28]. After the balloon is removed at the six month time point, there is some weight regain but at 12 months there is still more weight lost by the 'balloon' group (some 9% of initial weight), whilst those in the control group maintain their weight loss. There are side effects of the balloon, especially cramping and vomiting, but those treated this way have a significant increase in their measured quality of life. Other devices being investigated include endoduodenal sleeves (a plastic sleeve which lines the duodenum) and gastric pacemakers. These too need to be placed endoscopically.

## General issues

Obviously many of those overweight or obese will already have an obesity-related comorbidity, either metabolic or mechanical (these are discussed elsewhere in this volume). These problems should be treated actively, but at the same time a weight-loss program should be instituted and conversely during a weight-loss program there should be active, effective treatment of such comorbidities. Weight loss can help improve these medical problems [29] and also reduce the medications required to treat them [30], as well as improving quality of life.

At the same time care should be taken to not use, or minimise the use and dose of, drugs which may cause weight gain. These include antipsychotics and antidepressants, steroid therapy, anticonvulsants (especially valproate) and medications used to manage diabetes. Where possible an alternate drug which has no effect on weight, or which can help weight loss should be chosen. For example, use of an SSRI such as fluoxetine in depression can promote weight loss, topiramate is an anti-epileptic which promotes weight loss [31], metformin may produce or assist with weight loss and some of the newer anti-diabetic agents, such as the GLP1 agonists, promote weight loss.

## Maintenance of weight loss

This is a vexed issue. Within three to five years of completed and effective weight-loss programs most of the weight loss will have been regained, though there is increasing evidence these programs can be effective [32]. Even following bariatric surgery, where there is substantial weight loss which can be maintained for years, there tends to be a slow regain (though not to initial weight). This regain is due to 'relaxation' of the lifestyle program and is caused by underlying physiological drivers, such as elevated appetite hormones causing increased hunger, which force a reversion to old eating patterns (as an example).

There are some individuals who lose weight and do keep it off for years. These individuals undertake high levels of physical activity, eat breakfast and regular meals, a diet that is low in fat, and they self monitor which is a way of catching 'slips' early and correcting them. However, this type of response is not the common one.

Does that mean weight-loss programs are always failures and therefore should not really be attempted? The answer to this is a resounding 'no' on at least two counts. Firstly, weight loss does have an effect on many aspects: the control and prevention of disease, greater mobility, better quality of life and so on. There is even evidence from the diabetes prevention trials that a beneficial metabolic effect, the so-called metabolic memory, persists for years after the program has ceased and even if weight is regained.

Secondly, most of the reported weight-loss 'failures' are the follow-up of a single intervention. It is important that it is realised that the struggle against the issue of excess weight is a chronic, lifelong one. Programs need to be developed which include regular follow-up, which use pharmacotherapy to maintain loss, and have acute interventions if there is a small amount of regain (in our clinic we use a regain of three kilograms as the trigger for a

more intense effort). The US-based Look AHEAD program in people with type 2 diabetes is one such example [32]. Multidisciplinary chronic care programs will become even more necessary as the cohort of those who have had bariatric surgery increases – this group will need such lifetime follow-up. With the development of these long-term programs there must be research on effectiveness and over time, long-term weight loss and maintenance will be achieved.

Management of obesity in children and adolescents: whom and when to treat?

#### *Presentations to healthcare settings*

Children and adolescents who are overweight or obese attend both primary and tertiary healthcare settings more frequently than might be expected from the background prevalence of this condition [33–36]. This is probably because of the health complications linked to their weight status. However, they rarely present to the doctor specifically for the problem of obesity. In a large random sample of Australian general practice surgeries, the BEACH study, 29.6% of two- to 18-year-olds were overweight or obese (11.4% overall were obese), compared with a background population prevalence of 23%–25% (5%–8% obese). Unfortunately, these patients are unlikely to have the problem of obesity addressed by the clinician. In the BEACH study, only 1:60 of those who were overweight or obese had any form of management provided for this issue [34].

Why might this situation be? While Australian general practitioners (GPs) state that they are committed to dealing with the consequences of obesity, they also cite a range of barriers to managing it, including inadequate time and reimbursement, an apparent lack of effective interventions and support services, low levels of parent or patient motivation, the complexity and sensitivity of the issue, and the need for further training [37]. Families themselves are somewhat circumspect about the role of GPs on this issue and do not see GPs as the primary source of advice about management of obesity in young people [38].

#### *Indicators for treatment*

So, should children and adolescents affected by obesity seek or receive treatment, and, if so, where? As a general principle, assessment and treatment is especially warranted for the following individuals [39–42]:

- Those who are moderately to severely obese
- Adolescents
- Those with obesity-associated complications, such as insulin resistance, obstructive sleep apnoea, psychosocial distress, fatty liver disease, cardio-metabolic risk factors, or orthopaedic complications
- Those with a high risk family history, such as parents, grandparents, siblings or aunts/uncles with type 2 diabetes, premature heart disease, a history of bariatric surgery, obstructive sleep apnoea, hypertension or dyslipidaemia
- Those from a high-risk ethnic group, where cardio-metabolic complications are more

common, such as Aboriginal and Torres Strait Islanders, Maori and Pacific people, those from Mediterranean or Middle-Eastern countries or the Indian subcontinent, and native Americans.

#### *Where should treatment be provided?*

Given the high prevalence of child and adolescent obesity in most Westernised, and rapidly Westernising, countries, and its chronicity, coordinated models of care for health service delivery are needed. However, no country has yet established a cost-effective model of care for obesity management. One potential approach is that adapted from the UK National Health Service [43] and Kaiser Permanente [44] chronic disease pyramid of care. This is based upon a tiered level of service delivery relating to severity of disease.

Ideally, the vast majority of those affected by the problem of obesity should be managed via self-care or family-based care, with support from GPs or community-based health service providers. Such patients should be offered healthy lifestyle counselling or community-based group programs. The next stage of management involves more targeted recommendations for behavioural change, supported by involvement of allied health professionals, such as dietitians, or, indeed, a multidisciplinary team of therapists. The final stage, which is offered to children and adolescents with severe obesity, is provided by a specialist team and may include provision of pharmacotherapy, very low energy diets, or even bariatric surgery. The patient's age, severity of obesity and obesity-associated complications, and level of engagement with, and success of, previous interventions, should determine which stage of therapy is offered.

Unfortunately, such a staged approach to care is yet to be resourced or implemented widely in countries such as Australia. In reality, most children and adolescents who are affected by obesity will be managed by their GP, possibly with some involvement of a practice nurse or dietitian. Certainly, those children or adolescents who have severe obesity should be assessed, and have their care coordinated, by a paediatrician or adolescent physician, ideally with involvement of a multidisciplinary team of other therapists.

#### *Lifestyle treatment*

The mainstay of treatment of obesity in this age-group, as in adults, is lifestyle change. What is the evidence for this?

The 2009 Cochrane review on the treatment of child and adolescent obesity included 64 randomised controlled trials [45]. No one specific treatment program was recommended over another; however, positive outcomes were identified in several studies. A meta-analysis of several studies showed that family-targeted lifestyle intervention, involving various combinations of dietary, physical activity and behaviour modification, led to a significantly greater reduction in BMI or BMI z-score (ie BMI adjusted for age and sex) at six months than 'standard care'. For children aged less than 12 years there was a modest average reduction of 0.06 in BMI z-score, and in adolescents there was an average reduction of just over three BMI units.

However, many published studies are limited by varying attrition rates, small sample sizes, different measures of change in body size, and lack of assessment of other outcomes (for example, broader medical, psychosocial and behavioural outcomes). And they have typically involved fairly homogeneous patient samples managed in a tertiary care setting. Although recent trials are addressing some of these issues, much of the evidence to support effective intervention may not be readily generalisable to other clinical settings. For example, 'real-world' obesity clinics are usually less well resourced than programs in clinical trials, and patients seen in such clinics may be quite different from those who volunteer for trials. They may have more social disadvantage, or have psychosocial complications and other problems that make adherence to treatment more difficult. Not surprisingly, therefore, studies in clinical practice have demonstrated poorer results than in formal clinical trials [46]. Nevertheless, the broad principles of management are well recognised and are outlined in Box 1.

**Box 1. Principles of obesity management in children and adolescents**

- management of obesity-associated comorbidities
- family involvement
- a developmentally appropriate approach
- long-term behaviour modification
- dietary change
- increased physical activity
- decreased sedentary behaviours
- consideration of the use of pharmacotherapy and other forms of non-conventional therapy
- plan for longer-term weight maintenance strategies.

Effective management of obesity-associated comorbidities, such as sleep apnoea, dyslipidaemia, hypertension, non-alcoholic fatty liver disease or type 2 diabetes mellitus, is vital. Ideally, patients should be co-managed, in a coordinated way, by relevant specialist teams. In all cases, effective weight management should be a key element of the treatment of the comorbidity. Further discussion of the management of these comorbidities is beyond the scope of this review.

*Family focus*

Many clinical trials show that family-based interventions can lead to long-term relative weight loss (that is, from two to ten years) [45, 47–50]. Parental involvement is vital when managing obese younger children in particular [45, 48, 51–53]. This makes sense, as parents are normally the people in the family who are responsible for the food and physical activity environment, such as buying and cooking food, the types of routines around meals and meal times, rules around TV viewing and bed-time, role-modelling of eating and activity behaviours, and the use of the family car.

### *A developmentally appropriate approach*

The developmental age of the patient – whether they are pre-adolescent, or adolescent, or, indeed, whether they have a developmental disability – will influence whether the patient per se is involved in treatment, and the type of parental engagement. A different approach is usually needed for pre-adolescent children compared to adolescents.

### **Pre-adolescent children**

When managing pre-adolescent children, a parent-focused intervention, without direct engagement of the child, appears to have better outcomes to a child-centred approach [50, 54]. Obese children aged six to 11 years and their parents were randomly assigned to either parent-only group sessions (with an emphasis on general parenting skills), or child-only group sessions. At one and seven years follow-up from baseline, a significant reduction in overweight was observed in the parent-only group compared with the child-only group. At one year, there was a 14.6% reduction in overweight in the parent-only group versus 8.1% in the child-only group ( $p < 0.03$ ), with a nine times greater dropout rate in the child-only group [50]. At the seven-year follow-up, 60% of the children in the parent-only group, versus 31% of those in the child-only group, were classified as non-obese [54]. These and other studies suggest that when treating pre-adolescent children with obesity, therapy sessions involving the parent or parents alone, without the young child being present, could be the most effective.

### **Adolescents**

There are fewer studies looking specifically at treatment of adolescents affected by obesity. The previously mentioned 2009 Cochrane review included 27 trials involving participants aged 12 years and older [45]. Twelve studies used behavioural lifestyle modification, ten involved drug therapy, three used physical activity, and two involved diet. Most included studies were of high intensity and were offered in secondary or tertiary level care settings. Many offered separate sessions for adolescents and parents. In clinical practice, it may be useful to have at least some of the session time just involving the adolescent and therapist, engaging the parent later.

### Behaviour modification

Behaviour modification in weight management includes a set of techniques employed to change thought processes and actions associated with eating, physical activity and sedentary behaviours [55]. Many trials of obesity management in children and adolescents have included a behavioural component, although these are often poorly described. In general, the greater the range of behaviour change techniques used, the better the weight outcomes [48]. Goal setting, stimulus control and self-monitoring are three key behaviour change techniques used.

Goal setting can include performance goals (such as changing eating or activity behaviours) or outcome goals (such as specific weight loss). An example of a well-specified goal is:

I will not buy any biscuits, chocolates or other high-fat foods during the weekly shopping. In order to make this easier, I will leave the children at home and shop on my own. If the children ask for junk food, then I will offer yoghurt or fruit instead.

Considerable session time may be required to set and review behaviour change strategies with families and young people [56].

Stimulus control refers to modifying or restricting environmental influences to aid weight control. The broader environment, whether at home, school or beyond, has a profound influence on a person's decisions around eating, physical activity or sedentary behaviours. Although many of these environmental influences are beyond the scope of an individual or family to change (for example, food marketing or food pricing), many opportunities exist for families to implement stimulus control in the everyday environment. Examples include: not eating in front of the television; not having television or other screens in bedrooms; using smaller plates, bowls and spoons; and not storing unhealthy food choices in the house [47].

Self-monitoring is the detailed recording of a specific behaviour and can contribute to better weight control in children and adolescents [57]. This self-monitoring can take several forms: use of a food diary, television use diary, daily pedometer measurement of physical activity, or weekly weighing, as examples [58].

Motivational interviewing is a 'client-centred, directive method for enhancing intrinsic motivation to change by exploring and resolving ambivalence' that is increasingly being used in obesity management [59]. It requires an empathetic and collaborative therapist who responds to the patient's (or parent's) ambivalence to change in a nonjudgmental fashion. In addition, the therapist uses open-ended questions and reflective listening techniques to help direct communication toward change in behaviour [41, 59, 60].

#### Dietary change and eating behaviours

A 2006 systematic review of paediatric obesity trials showed that interventions containing a dietary component were effective in achieving relative weight loss [61]. The Traffic Light (also known as the Stop Light) diet was the most commonly used diet intervention. Briefly, in the Traffic Light diet, foods are colour-coded to indicate those to be eaten freely (green) and those to be eaten more cautiously (amber and especially red) [55]. Although food or kilojoule exchange programs were also identified as common dietary interventions in the above review, only one trial compared diets of varying macronutrient composition. Since then, a substantial body of evidence has accumulated on the effects of diets with different macronutrient profiles on weight loss or maintenance, especially in adults, with results tending to favour diets proportionately higher in protein relative to carbohydrate and with a low glycaemic index [62–66]. For example, results published in 2010 from the DiOGenes randomised controlled trial compared the effect of five *ad libitum* diets (with variations in glycaemic index and protein content) on body composition in European children: a low protein–high glycaemic index diet increased body fat, whereas overweight or obesity decreased in the high protein–low glycaemic index diet group [65].



What recommendations should currently be given? Dietary interventions should follow national nutrition guidelines and have an emphasis on the following [47, 52, 66–68]:

- regular meals
- eating together as a family
- choosing nutrient-rich foods that are lower in energy and glycaemic index
- increased vegetable and fruit intake
- healthier snack food options
- decreased portion sizes
- promotion of water as the main beverage, and a reduction in sugary drink intake
- involvement of the entire family in making the change to a sustainable and healthy food intake.

In advising patients and families on dietary change, is there a potential risk of an eating disorder developing? Although most people with obesity do not have binge-eating disorder, the more severe the obesity, the more likely the patient is to have binge-eating disorder [69]. In addition, obesity in childhood, or parental obesity, is a risk factor for later bulimia, and overweight adolescents are more likely to use a range of unhealthy behaviours to binge-eat [70]. On the other hand, evidence indicates that professionally run paediatric obesity programs do not increase the risk of disordered eating and might, indeed, improve psychological wellbeing [71]. For these reasons, Hill has highlighted the need for ‘properly and expertly managed’ weight-control interventions to avoid the risk of an eating disorder [72].

### Physical activity and sedentary behaviours

#### *Physical activity*

Involvement of children in a lifestyle program (for example, walking, running, cycling or swimming, based on the family’s preference) leads to greater reductions in percentage overweight at six months and 17 months when compared with a program of organised aerobic exercise involving the same prescribed level of energy expenditure [73]. A study of similar design, but including a third control group involved in calisthenics, and with follow-up for ten years, showed that the lifestyle and aerobic exercise programs were superior in terms of percentage overweight reduction to the calisthenics control group [74].

A systematic review and meta-analysis of exercise interventions in overweight children and adolescents indicated that 155 to 180 minutes per week of supervised moderate-to-high intensity physical activity (with or without an associated dietary intervention) was effective in reducing body fat, although the effects on body weight and abdominal fat were inconclusive [75]. In 2008, the effect of resistance training on metabolic fitness in children and adolescents was investigated in a systematic review of 12 trials, with eight of them targeting people who were overweight or obese [76]. Most of the interventions

included in that review involved circuit-type resistance training (moderate–high velocity, low–moderate load) involving machine weights. Unfortunately, limitations in study design and reporting prohibited definitive conclusions being established, but the beneficial effect of resistance training on health outcomes in adults was noted by the study authors.

### *Sedentary behaviours*

Several studies and clinical guidelines address the issue of targeting sedentary behaviours. For example, in one study, 90 families of children aged eight to 12 years with obesity were assigned to different arms of a behavioural weight-control program in which either sedentary behaviours or physical activity were targeted, with two different levels of behaviour change being required. At two-year follow-up, similar improvements in aerobic fitness, body-fat percentage and percentage overweight were observed for all treatment arms, indicating that modifying sedentary behaviour is as effective as changing physical activity levels [77].

What recommendations can be given in terms of physical activity and reduction of sedentary behaviour? [39, 41–43, 45, 68]

- Increased physical activity may best result from a change in incidental or unplanned activity; for example, walking or cycling for transport, household chores, and playing with friends or family.
- Organised exercise programs, such as playing sports, are also important.
- Encourage children or adolescents to choose activities that they enjoy as these activities are more likely to be sustainable.
- Limit television and other ‘small screen’ recreational viewing to less than two hours per day. This may be extremely challenging given the multiple types of screen-based activities available to young people.
- Parental involvement is crucial if an increase in physical activity or a decrease in sedentary behaviour is to occur, including monitoring and limiting television use, role-modelling of healthy behaviours, and providing access to recreation areas or recreational equipment.

### The Children’s Hospital Big Five

At the Children’s Hospital at Westmead, in Sydney, five strategies are emphasised when initially managing families with obese children or adolescents:

- Eat breakfast
- Choose water as your main drink
- Eat together as a family once a day, without the TV being on
- Limit TV and other ‘small screens’ to less than two hours per day
- Play, or be active, outside for at least 60 minutes each day.

### Adjunctive therapy

The evidence to guide the use of less orthodox treatment approaches – such as VLEDs, pharmacological therapy or bariatric surgery – in the treatment of severe obesity is more limited than for behavioural interventions. In general, such therapies should occur on the background of a behavioural weight-management program and be restricted to specialist centres with expertise in managing severe obesity.

#### *Very low energy diets (VLEDs)*

As noted above, in adults, VLEDs are used to achieve rapid weight-loss prior to, or in conjunction with, the use of other longer-term treatment interventions [78]. VLEDs can also be associated with safe and substantial rapid weight loss in adolescents, typically producing six to 15 kg weight loss over three to 12 weeks [79–81]. However, most studies have been based on small sample sizes (less than 20 participants) and have lacked a control group, with very few studies having long-term follow-up [82]. To date, no randomised controlled trials have examined the effectiveness of a weight-management program incorporating initial VLED treatment in obese adolescents. The US Expert Committee recommended that VLEDs should ideally be used with severely obese patients who are managed by a multidisciplinary team in a tertiary care setting. They are not appropriate for use in young children.

#### *Pharmacology in the treatment of childhood and adolescent obesity*

The use of most anti-obesity agents (whether previously or currently available, or in development) is off label for children and adolescents. These agents should not be used in children and younger adolescents, as the risk outweighs the benefit and weight management depends primarily on parental control of the environment. There will be exceptional cases when anti-obesity agents might be trialled, with the appropriate consents in place. This will usually be for secondary obesity, for example hypothalamic hyperphagia syndromes. Somatostatin analogues have also been used with some success in children in this situation [83].

The choice of anti-obesity agents is limited and these should be prescribed as part of a lifestyle intervention, with diet and activity modification and behavioural intervention [84]. These agents may also be prescribed as part of bariatric surgery considerations and to enhance weight maintenance after weight loss in the older adolescent. As with adults, adolescents are likely to have unrealistic expectations of the benefits of drug therapy and these expectations may be greater in mid-adolescence when cognitive developmental stage limits their ability to understand the degree of weight loss possible. Adolescents generally have a very low tolerance for side effects and are likely to discontinue medication for tenuous reasons, including the lack of instantaneous benefit.

There is some data on orlistat use in adolescents with greater weight loss compared to placebo [84, 85]. The side effects of flatulence, diarrhoea with urgency and faecal leakage are highly socially distressing and for most adolescents unacceptable. If they are prescribed orlistat they will simply not take it when they have a fatty meal away from home, which

may be when most higher calorie meals are consumed. For those who persist, careful monitoring of, and supplementation of, fat-soluble vitamins is important as adolescents appear more prone to deficiencies than adults.

Phentermine remains available in some jurisdictions and also for use in those over 12 years old. It has adverse effects including palpitations, headaches, hypertension, insomnia and euphoric states. Phentermine also has the potential for abuse and should not be used in adolescents, many of whom already have binge-eating disorders and will be seeking medication to produce some anorexia. As depression is often present in this situation, fluoxetine might be beneficial as a centrally acting psychotropic which does not induce weight gain.

There is some evidence in children and adolescents that metformin may have a beneficial effect on body weight. It is the drug of choice in those with type 2 diabetes, who are invariably overweight, as it does not induce further weight gain. There is some evidence from small trials that metformin improves metabolic profile and reduces visceral fat in clinical scenarios where there is insulin resistance and weight gain, but not overt diabetes. These trials include the use of atypical anti-psychotics, such as risperidone, which are frequently prescribed for behavioural disturbances [86], and in medical conditions where physical activity is severely restricted [87]. The actual weight-loss results are less consistent. Metformin, also off label, may be advantageous in the obese adolescent female with the polycystic ovary syndrome and anovulatory bleeding or acanthosis nigricans. Both these conditions will improve and in some there may be a weight benefit, based on some small clinical trials. It is essential that a low start dose of metformin is chosen, 250 mg daily commencing with the evening meal. The dose should be built up to two grams over three to four weeks to minimise risk of the well-known side effects of abdominal pain and diarrhoea. Vitamin B12 levels should be monitored as the development of deficiency is not uncommon.

Puberty is a time where weight gain can accelerate in an overweight child or adolescent [88]. While it is clear from the previous discussion that there are no available pharmacotherapeutic agents to counter this, clinicians should carefully consider prescription of commonly used drugs which may further increase weight. Select if possible an oral contraceptive which contains one of the newer gestagens, such as desogestrel or drospirenone. Consider etonogestrel, rather than medroxyprogesterone acetate as a long-acting contraceptive preparation.

### *Bariatric surgery*

Bariatric surgery is a well-recognised form of therapy for adults with severe obesity, especially if medical therapy has failed [89]. What about its role in adolescents? Almost all of the literature in this area is of case studies or case series, with only one published randomised controlled trial to date [90].

*Evidence for bariatric surgery in adolescents*

A 2008 systematic review of bariatric surgery for adolescent obesity included studies that reported outcome data for patients under the age of 21 years (average age 16.8 years, range nine to 21) who were followed up for at least 12 months [91]. Nineteen papers were included, including eight studies of laparoscopic gastric banding, six studies of Roux-en-Y gastric bypass, and five studies of other surgical procedures. The laparoscopic adjustable banding procedures, with one to three years follow-up, had 95% confidence intervals for weight loss of  $-13.7$  to  $-10.6$  BMI units. No perioperative deaths were reported, and the main complication reported was the need for re-operation (for band slippage), which occurred in 8% of patients. For Roux-en-Y gastric bypass procedures, the mean weight loss was greater, with 95% confidence intervals of  $-17.8$  to  $-22.3$  BMI units, albeit over a longer period of follow-up (as the procedure has been established for longer). Some deaths were reported after this type of surgery. The most frequently reported complications related to protein-energy malnutrition and micronutrient deficiency. Note that none of the included studies in this systematic review was a randomised controlled trial, and hence the strength of evidence could only be classed as moderate to weak. However, these findings are similar to those seen in comparisons of bariatric surgery in adults [89].

After this review was published, the results of the first randomised controlled trial of bariatric surgery in adolescents with severe obesity became available in 2010 [90]. This study was undertaken in Melbourne and included 50 adolescents with severe obesity (BMI  $>35$  kg/m<sup>2</sup>) aged between 14 and 18 years who were randomly assigned to receive either a supervised lifestyle intervention, involving reduced energy intake, increased physical activity and behaviour modification, or laparoscopic adjustable gastric banding. By 24 months, the surgical group had an average reduction in BMI of 12.7 units, versus 1.3 units in the lifestyle group. The surgical group also had marked improvements in both cardiometabolic status and quality of life. However, eight operations (33%) were required in seven patients for revisional procedures.

*Guidelines on bariatric surgery*

In 2009, Australian and New Zealand consensus recommendations for bariatric surgery in adolescents were published by the professional bodies representing paediatricians, paediatric surgeons and bariatric surgeons [92]. The recommendations included the following:

- Patient: A minimum age of 15 years (or 14 years in exceptional circumstances); post-pubertal; presence of severe obesity (a BMI  $>40$  kg/m<sup>2</sup>, although it should be considered in adolescents with a BMI  $>35$  kg/m<sup>2</sup> in the presence of severe obesity-associated complications); presence of an associated severe comorbidity; persistence of obesity despite involvement in a formal multidisciplinary and supervised program of lifestyle modification and pharmacotherapy; the adolescent and family understand, and are motivated to participate in, the ongoing treatment, lifestyle change and review following surgery; the adolescent is able to provide informed consent for the surgery.

- Surgical expertise and facilities: Surgery should be performed by an experienced bariatric surgeon affiliated with a team experienced in the assessment and long-term follow-up of the metabolic and psychosocial needs of the adolescent bariatric patient and family; the working party strongly recommended that publicly funded bariatric surgery be made available to those in need.
- Type of surgery: The majority recommendation was that the primary bariatric surgical procedure of choice for adolescents in Australia and New Zealand is laparoscopic adjustable gastric banding as it has good weight-based outcomes, has a low complication rate and is potentially reversible.
- Post-operative management and follow-up: Patients should be managed in the immediate post-operative period by a surgeon and bariatric surgical team with experience in adolescent care; adolescents will need more frequent post-operative follow-up than adult patients (eg four to six on weekly basis initially); patients will need ongoing care by a multidisciplinary team; issues such as improved fertility following weight loss, and hence the need for contraception, need to be considered; follow-up needs to extend beyond ten years, and ideally for the whole life.

One of the major challenges in Australia, as elsewhere, is how to ensure equitable access to such services for affected adolescents.

#### Long-term weight maintenance

Few high-quality studies have reported long-term outcomes of treatment of childhood and adolescent obesity [45]. In those which have reported long-term outcomes, a high proportion of participants maintained a reduction in overweight from two to ten years from baseline without additional intervention after the initial treatment phase (weeks to months) [45, 67, 93, 94]. However, in those who have undergone initial weight-management intervention, a period of further therapeutic contact (varying from four to 12 months) seems to slow weight regain [95]. At present, there is only limited evidence to guide the nature and type of weight-maintenance interventions in the child and adolescent age group.

#### Conclusions

Obesity and overweight are common problems at most life stages. They produce many associated comorbidities and psychosocial issues contributing to substantial public health problems and costs.

Treatment can be, and is effective. A lifestyle program should be associated closely with the treatment. Such a multidisciplinary program includes appropriate healthy eating, increased activity (both planned and incidental), behavioural techniques and counselling to help overcome ingrained habits and issues which may lead to weight gain. For children, the involvement of family in treatment is absolutely necessary. Finally, the program should not just be a 'once-off' because weight is regained due to physiological systems which drive appetite after weight loss. The program should be reinstated or reinforced at regular intervals and patients given a weight regain limit at which to return for a 'booster' program.

A single treatment may be effective for those who need to lose a few kilos (5% or so of their initial weight), but for those with higher grades of obesity or complications of obesity, appropriate adjunctive therapies should be utilised and consideration given, when necessary, to bariatric surgery.

## References

1. Caterson I (2009). Medical management of obesity and its complications. *Annals Academy of Medicine Singapore*, 38: 22–27.
2. Bjorvell H & Rossner S (1992). A ten-year follow-up of weight change in severely obese subjects treated in a combined behavioural modification programme. *International Journal of Obesity*, 16(8): 623–25.
3. Jebb S, Ahern A, Olson A, Aston L, Holzapfel C, Stoll J, et al. (2011). Primary care referral to a commercial provider for weight loss treatment, relative to standard care: an international randomised controlled trial. *The Lancet*, 378(9801): 1485–92.
4. Sacks F, Bray G, Carey V, Smith S, Ryan D, Anton S, et al. (2009). Comparison of weight-loss diets with different compositions of fat, protein and carbohydrates. *New England Journal of Medicine*, 360: 859–73.
5. McMillan-Price J, Petocz P, Atkinson F, O'Neill K, Samman S, Steinbeck K, et al. (2006). Comparison of 4 diets of varying glycemic load on weight loss and cardiovascular risk reduction in overweight and obese young adults: a randomized controlled trial. *Archives of Internal Medicine*, 166: 1466–75.
6. Dansinger ML, Gleason JL, Griffith JL, Selker HP & Schaefer EJ (2005). Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *Journal of the American Medical Association*, 293(1): 43–53.
7. Despres JP, Tremblay A, Nadeau A & Bouchard C (1988). Physical training and changes in regional adipose tissue distribution. *Acta Medica Scandinavica, Supplementum*, 723: 205–12.
8. Despres JP & Lamarche B (1993). Effects of diet and physical activity on adiposity and body fat distribution: implications for the prevention of cardiovascular disease. *Nutrition Research Reviews*, 6(1): 137–59.
9. Despres J-P, Pouliot M-C, Moorjani S, Nadeau A, Tremblay A, Lupien PJ, et al. (1991). Loss of abdominal fat and metabolic response to exercise training in obese women. *American Journal of Physiology*, 261(2): E159–E67.
10. Wadden TA & Clark VL (2005). Behavioural treatment of obesity: achievements and challenges. In PG Kopelman, ID Caterson & WH Dietz (Eds). *Clinical obesity in adults and children*. (pp350–62). 2nd edn. Malden Oxford Carlton: Blackwell.
11. Richman RM, Steinbeck KS & Caterson ID (1992). Severe obesity: the use of very low energy diets or standard kilojoule restriction. *Medical Journal of Australia*, 156(11): 768–70.
12. Flechtner-Mors M, Ditschuneit HH, Johnson TD, Suchard MA & Adler G (2000). Metabolic and

weight loss effects of long-term dietary intervention in obese patients: four-year results. *Obesity Research*, 8(5): 399–402.

13. Lau N & Caterson I (2011). Meal replacement products and very low calorie diets in adult obesity. *Royal College of Pathologists Bulletin*, 155: (172–74).
14. Dixon J, O'Brien PE, Playfair J, Chapman L, Schachter LM, Skinner S, et al. (2008). Adjustable gastric banding and conventional therapy for type 2 diabetes: a randomized controlled trial. *Journal of American Medical Association*, 299(3): 316–23.
15. Lean M & Finer N (2006). ABC of obesity. Management: Part II – Drugs. *British Medical Journal*, 333(7572): 794–97.
16. Padwal R & Majumdar S (2007). Drug treatments for obesity: orlistat, sibutramine, and rimonabant. *The Lancet*, 369(9555): 71–77.
17. James W, Caterson I, Coutinho W, Finer N, Van Gaal L, Maggioni A, et al. (2010). Effect of sibutramine on cardiovascular outcomes in overweight and obese subjects. *New England Journal of Medicine*, 363(10): 309–17.
18. Swinburn BA, Carey D, Hills AP, Hooper M, Marks S, Proietto J, et al. (2005). Effect of orlistat on cardiovascular disease in obese adults. *Diabetes Obesity and Metabolism*, 7(3): 254–62.
19. Caterson ID (1988). Overweight, obesity and the role of the pharmacist. *Australian Pharmacist*, 7: 13–16.
20. Wittert G, Caterson I & Finer N (2007). The clinical effectiveness of weight loss drugs. *Journal of Obesity Research and Clinical Practice*, 1(1): 1–5.
21. Astrup A, Carraro R, Finer N, Harper A, Kunesova M, Lean M, et al. (2011). Safety, tolerability and sustained weight loss over 2 years with the once-daily human GLP-1 analog, liraglutide. *International Journal of Obesity*, Advance online publication, 16 August 2011.
22. Gadde K & Allison D (2009). Combination therapy for obesity and metabolic disease. *Current Opinion in Endocrinology, Diabetes and Obesity*, 16(5): 353–58.
23. Greenway F, Fujioka K, Plodkowski R, Mudaliar S, Guttadauria M, Erickson J, et al. (2010). Effect of naltrexone plus bupropion on weight loss in overweight and obese adults (COR-1): a multicentre, randomised, double-blind, placebo-controlled, phase 3 trial. *The Lancet*, 376(9741): 595–605.
24. Sjostrom L, Narbro K, Sjostrom C, Karason K, Larsson B, Wedel H, et al. (2007). Effects of bariatric surgery on mortality in Swedish obese subjects. *New England Journal of Medicine*, 357: 741–52.
25. Panel IDFC (2011). Bariatric surgical and procedural interventions in the treatment of obese patients with type 2 diabetes: a position statement from the International Diabetes Federation Taskforce on Epidemiology and Prevention. September [Online]. Available: [www.idf.org/webdata/Bariatric-Surgery-Press-Briefing.pdf](http://www.idf.org/webdata/Bariatric-Surgery-Press-Briefing.pdf) [Accessed 24 October 2011].
26. Padwal R, Klarenbach S, Wiebe N, Birch D, Karmali S, Manns B, et al. (2011). Bariatric surgery: a systematic review and network meta-analysis of randomized trials. *Obesity Reviews*, 12: 602–21.
27. Tice J, Karliner L, Walsh J, Petersen A & Feldman M (2008). Gastric banding or bypass? A systematic review comparing the two most popular bariatric procedures. *American Journal of Medicine*, 121: 885–93.



28. Fuller N, Pearson S, Lau N, Markovic T, Steinbeck K, Chettiar R, et al. (2010). A prospective, randomized, controlled trial of the bioenterics intragastric balloon (BIB) in the treatment of obese individuals with the metabolic syndrome. *Obesity Reviews*, 11(Suppl. 1): 436.
29. Unick J, Beavers D, Jakicic J, Kitabachi A, Knowler W, Wadden T, et al. (2011). Effectiveness of lifestyle interventions with severe obesity and type 2 diabetes: results from the Look AHEAD trial. *Diabetes Care*: (Aug 11) epub ahead of print.
30. Group TLAR (2007). Reduction in weight and cardiovascular disease risk factors in individuals with type 2 diabetes: one-year results of the look AHEAD trial. *Diabetes Care*, 30: 1374–83.
31. Astrup A, Caterson I, Zelissen P, Guy-Grand B, Carruba M, Levy B, et al. (2004). Topiramate for long-term weight maintenance of weight loss induced by a low-calorie diet in obese subjects. *Obesity Research*, 12: 1658–69.
32. Wing R & Group LAR (2010). Long-term effects of a lifestyle intervention on weight and cardiovascular risk factors in individuals with type 2 diabetes mellitus: four-year results of the Look AHEAD trial. *Archives of Internal Medicine*, 170: 1566–75.
33. Benson L, Baer H & Kaelber D (2007). Trends in the diagnosis in overweight and obesity in children and adolescents: 1999–2007. *Pediatrics*, 123: e153–e8.
34. Cretikos M, Valenti L, Britt H & Baur L (2008). General practice management of overweight and obesity in children and adolescents in Australia. *Medical Care*, 4: 1163–69.
35. O'Connor J, Youde L, Allen J & Baur L (2004). Obesity and under-nutrition in a tertiary paediatric hospital. *Journal of Paediatrics and Child Health*, 40: 299–304.
36. Woo J, Zeller M, Wilson K & Inge T (2009). Obesity identified by discharge ICD-9 codes underestimates the true prevalence of obesity in hospitalised children. *Journal of Paediatrics*, 154: 327–31.
37. King L, Loss J, Wikenfeld, Pagnini D, Booth M & Booth S (2007). Australian GPs' perceptions about child and adolescent overweight and obesity: the Weight of Opinion study. *British Journal of General Practice*, 57: 124–29.
38. Shrewsbury V, King L, Hattersley L, Howlett S, Hardy L & Baur L (2010). Adolescent-parent interactions and communication preferences regarding body weight and weight management: a qualitative study. *The International Society of Behavioral Nutrition and Physical Activity*, 7: 16.
39. Barlow S & The Expert Committee (2007). Expert Committee recommendations regarding the prevention, assessment, and treatment of children and adolescent overweight and obesity: summary report. *Pediatrics*, 120(Suppl. 4): S164–S92.
40. Krebs N, Himes J, Jacobson D, Nicklas T, Guilday P & Styne D (2007). Assessment of child and adolescent overweight and obesity. *Pediatrics*, 120(Suppl. 4): S193–S228.
41. National Health and Medical Research Council (2003). Clinical practice guidelines for the management of overweight and obesity in children and adolescents. Canberra: NHMRC.
42. Network SIG (2007). Management of obesity: a national clinical guideline. Edinburgh: SIGN.

43. National Institute for Health and Clinical Excellence (2010). Obesity: guidance on the prevention, identification, assessment and management of overweight and obesity in adults and children. *National Health Service National Institute for Health and Clinical Excellence* [Online]. Available: [guidance.nice.org.uk/CG43/NICEGuidance/pdf/English](http://guidance.nice.org.uk/CG43/NICEGuidance/pdf/English) [Accessed 2011].
44. Feacham R, Sekhri N & White K (2002). Getting more for their dollar: a comparison of the NHS with California's Kaiser Permanente. *British Medical Journal*, 324: 135–41.
45. Luttikhuis HO, Baur L, Jansen H, Shrewsbury V, O'Malley C, Stolk R, et al. (2009). Interventions for treating obesity in children. *Cochrane Database of Systematic Reviews*, 1.
46. Reinehr T, Widhalm K, L'Allemand D, Wiegand S, Wabitsch M, Holl R, et al. (2009). Two-year follow-up in 21,784 overweight children and adolescents with lifestyle intervention. *Obesity*, 17: 1196–99.
47. Dietz W & Robinson T (2005). Clinical practice: overweight children and adolescents. *New England Journal of Medicine*, 352: 2100–09.
48. McLean N, Griffin S, Toney K & Hardeman W (2003). Family involvement in weight control, weight maintenance and weight-loss interventions: a systematic review of randomised trials. *International Journal of Obesity*, 27: 987–1005.
49. Nuutinen O & Knip M (1992). Predictors of weight reduction in obese children. *European Journal of Clinical Nutrition*, 46: 785–94.
50. Golan M, Weizman A, Apter A & Fainaru M (1998). Parents as the exclusive agents of change in the treatment of childhood obesity. *American Journal of Clinical Nutrition*, 67: 1130–35.
51. Nowicka P & Flodmark C-E (2008). Family in pediatric obesity management: a literature review. *International Journal of Pediatric Obesity*, 3:(Suppl. 1): 44–50.
52. Young K, Northern J, Lister K, Drummond J & O'Brien W (2007). A meta-analysis of family-behavioural weight-loss treatments for children. *Clinical Psychology Reviews*, 27: 240–49.
53. Shrewsbury V, Steinbeck K, Torvaldsen S & Baur L (2011). The role of parents in pre-adolescent and adolescent overweight and obesity treatment: a systematic review of clinical recommendations. *Obesity Reviews*, 12(10): 759–69.
54. Golan M & Crow S (2004). Targeting parents exclusively in the treatment of childhood obesity: long-term results. *Obesity Research*, 12: 357–61.
55. Epstein L, Myers M, Raynor H & Saelens B (1998). Treatment of pediatric obesity. *Pediatrics*, 101(Suppl. 2): 554–70.
56. Brennan L, Walkley J, Lukeis S, Rsiteska A, Archer L, et al. (2009). A cognitive behavioural intervention for overweight and obese adolescents illustrated by four case studies. *Behaviour Change*, 26: 190–213.
57. Saelens B & McGrath A (2003). Self-monitoring adherence and adolescent weight control efficacy. *Child Health Care*, 32: 137–52.
58. Alm M, Neumark-Sztainer D, Story M & Boutelle K (2009). Self-weighing and weight control behaviors among adolescents with a history of overweight. *Journal of Adolescent Health*, 44: 424–30.

59. Miller W & Rollnick S (2002). *Motivational interviewing: preparing people for change*. 2nd edn. New York: Guildford Press.
60. Carels R, Darby L, Cacciapaglia H, Konrad K, Coit C, Harper J, et al. (2007). Using motivational interviewing as a supplement to obesity treatment: a stepped care approach. *Health Psychology*, 26: 369–74.
61. Collins C, Warren J, Neve M, McCoy P & Stokes B (2006). Measuring effectiveness of dietetic interventions in child obesity: a systematic review of randomised trials. *Archives of Pediatric and Adolescent Medicine*, 160(9): 906–22.
62. Abete I, Astrup A, Martinez J, Thorsdottir I & Zulet M (2010). Obesity and the metabolic syndrome: role of different dietary macronutrient distribution patterns and specific nutritional components on weight loss and maintenance. *Nutrition reviews*, 68(4): 214–31.
63. Hession M, Rolland C, Kulkarni U, Wise A & Broom J (2009). Systematic review of randomised controlled trials of low-carbohydrate vs. low fat/low-calorie diets in the management of obesity and its comorbidities. *Obesity Reviews*, 10(1): 36–50.
64. Thomas DE, Elliott EJ, Baur L (2007). Low glycaemic index or low glycaemic load diets for overweight and obesity Low glycaemic index or low glycaemic load diets for overweight and obesity. *Cochrane Database of Systematic Reviews*. Issue 3. Article number CD005105.
65. Papadaki A, Linardakis M, Larsen T, VanBaak M, Lindroos A, Pfeiffer A, et al. (2010). The effect of protein and glycemic index on children's body composition: the DIOGenes randomised study. *Pediatrics*, 126(5): E1143–E52.
66. Larsen T, Dalskov S, VanBaak M, Jebb S, Papadaki A, Pfeiffer A, et al. (2010). Diets with high or low protein content and glycemic index for weight loss maintenance. *New England Journal of Medicine*, 363(22): 2102–13.
67. Epstein L, Valoski A, Wing R & McCurley J (1990). Ten-year follow-up of behavioral, family based treatment for obese children. *Journal of the American Medical Association*, 264(19): 2519–23.
68. Whitaker R (2003). Obesity prevention in pediatric primary care: four behaviors to target. *Archives of Pediatric and Adolescent Medicine*, 157(8): 725–27.
69. Fairburn C, Welch S, Doll H, Davies B & O'Connor M (1997). Risk factors for bulimia nervosa: a community-based case-control study. *Archives of General Psychiatry*, 54(6): 509–17.
70. Patton C, Selzer R, Coffey C, Carlin J & Wolfe R (1999). Onset of adolescent eating disorders: population-based cohort study over 3 years. *British Medical Journal*, 318(7186): 765–68.
71. Butryn M & Wadden T (2005). Treatment of overweight in children and adolescents: does dieting increase the risk of eating disorders? *International Journal of Eating Disorders*, 37(4): 285–93.
72. Hill A (2007). Obesity and eating disorders. *Obesity Reviews*, 8(Suppl. 1): 151–55.
73. Epstein L, Wing R, Koeske R & Valoski A (1982). A comparison of lifestyle change and programmed exercise on weight and fitness in obese children. *Behavior Therapy*, 16(4): 345–56.
74. Epstein L, Valoski A, Wing R & McCurley J (1994). Ten year outcomes of behavioral family-based treatment for childhood obesity. *Health Psychology*, 13(5): 373–83.

75. Atlantis F, Barnes E & Singh M (2006). Efficacy of exercise for treating overweight in children and adolescents: a systematic review. *International Journal of Obesity*, 30(7): 1027–40.
76. Benson A, Torode M & FiararoneSingh M (2008). Effects of resistance training on metabolic fitness in children and adolescents: a systematic review. *Obesity Reviews*, 9(1): 43–66.
77. Epstein L, Paluch R, Gordy C & Dorn J (2000). Decreasing sedentary behaviors in treating pediatric obesity. *Archives of Pediatric and Adolescent Medicine*, 154(3): 220–26.
78. Tsai A & Wadden T (2005). Systematic review: an evaluation of major commercial weight loss programs in the United States. *Annals of Internal Medicine*, 142(1): 56–66.
79. Figueroa-Colon R, VonAlmen T, Franklin F, Schuftan C & Suskind R (1993). Comparison of two hypocaloric diets in obese children. *American Journal of Disease in Childhood*, 147(2): 160–66.
80. Suskind R, Sothorn M, Farris R, VonAlmen T, Schumacher H, Carlisle L, et al. (1993). Recent advances in the treatment of childhood obesity. *Annals of the New York Academy of Sciences*, 699: 181–99.
81. Widhalm K & Zwiauer K (1987). Metabolic effects of a very low calorie diet in obese children and adolescents with special reference to nitrogen balance. *Journal of the American College of Nutrition*, 6(6): 467–74.
82. Sothorn M, Udall J, Suskind R, Vargas A & Blecker U (2002). Weight loss and growth velocity in obese children after very low calorie diet, exercise and behavior modification. *Acta Paediatrica*, 89(9): 1036–43.
83. Tzotzas T, Papazisis K, Perros P & Krassas G (2008). Use of somatostatin analogues in obesity. *Drugs*, 68(14): 1963–73.
84. Greydanus D, Bricker L & Feucht C (2011). Pharmacotherapy for obese adolescents. *Pediatric Clinics of North America*, 58(1): 139–53.
85. Chanoine JP, Hampi S, Jensen C, Boldrin M & Hauptman J (2005). Effect of orlistat on weight and body composition in obese adolescents: a randomized controlled trial. *Journal of the American Medical Association*, 293(23): 2873–83.
86. Shin L, Bregman H, Breeze J, Noyes N & Frazier J (2009). Metformin for weight control in pediatric patients on atypical antipsychotic medication. *Journal of Child and Adolescent Psychopharmacology*, 19(3): 275–79.
87. Casteels K, Fieuws S, VanHelvoirt M, Verpoorten C, Goermans N, Coudyzer W, et al. (2010). Metformin therapy to reduce weight gain and visceral adiposity in children and adolescents with neurogenic or myogenic motor deficit. *Pediatric Diabetes*, 11(1): 61–69.
88. Jasik C & Lustig R (2008). Adolescent obesity and puberty: the ‘perfect storm’. *Annals of the New York Academy of Sciences*, 1135: 265–79.
89. Colquitt J, Picot J, Loveman E & Clegg A (2009). Surgery for obesity. *Cochrane Database of Systematic Reviews*, 2.
90. O’Brien P, Sawyer S, Laurie C, Brown W, Skinner S & Veit F (2010). Laparoscopic adjustable gastric

banding in severely obese adolescents: a randomized trial. *Journal of the American Medical Association*, 303(23): 519–26.

91. Treadwell J, Sun F & Schoelles K (2008). Systematic review and meta-analysis of bariatric surgery for pediatric obesity. *Annals Surgery*, 248(5): 763–76.

92. Baur L & Fitzgerald D (2010). Recommendations for bariatric surgery in adolescents in Australia and New Zealand. *Journal of Paediatrics and Child Health*, 46(12): 704–07.

93. Magarey A, Perry R, Baur L, Steinbeck K, Sawyer M, Hills A, et al. (2011). A parent-led family-focused treatment program for overweight 5–9 year olds: the PEACH RCT. *Pediatrics*, 127: 214–22.

94. Collins C, Okely A, Morgan P, Jones R, Burrows T, Cliff D, et al. (2011). Parent diet modification, child-centered activity or both in obese children: an RCT. *Pediatrics*, 127(4): 619–27.

95. Wilfley D, Stein R, Saelens B, Mockus D, Matt G, Hayden-Wade H, et al. (2007). Efficacy of maintenance treatment approaches for childhood overweight: a randomised controlled trial. *Journal of the American Medical Association*, 298(14): 1661–73.